7b and *8a* is more restricted. Closer examination revealed that *fzd7b* is expressed in the neural crest and the mesodermal core of the pharyngeal arches and in the chondrocytes of newly stacked craniofacial cartilage elements. The more restricted expression of *fzd7a* is found in the neural crest of the pharyngeal arches and later is limited to the mesenchymal tissues surrounding the craniofacial cartilage elements. In contrast, *fzd8a* is expressed in the pharyngeal endoderm. Our observations suggest a specific and at least partially non-redundant role of the examined *frizzled* genes. This characterization advances our knowledge of the role of Wnt signaling in craniofacial cartilage development and allows us to identify candidate Frizzleds for Wnt/PCP ligands during cartilage element elongation.

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Program/Abstract # 362
Loss of β-catenin in the Wnt1 expression domain results in an expansion of Rathke's pouch
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The anterior and intermediate lobes of the mouse pituitary gland arise from an invagination of oral ectoderm known as Rathke's pouch. The formation of Rathke's pouch occurs in response to signals emitted from the overlaying ventral diencephalon. A careful balance of the Wnt, Bmp, and Fgf signaling pathways in the ventral diencephalon is necessary to ensure the proper size and location of Rathke's pouch. Rathke's pouch may also be influenced by the surrounding mesenchyme, which is of both neural crest and head mesenchyme in origin. However, the role of mesenchyme in pituitary development is poorly understood. The *Wnt1-cre* is frequently used to conduct loss-of-function studies in the neural crest lineage. We have found that the loss of *β-catenin* in the *Wnt1* expression domain leads to a dramatic expansion of Rathke's pouch. This expansion is similar to those observed when the balance of signaling pathways within the ventral diencephalon is disrupted. While *Wnt1* is not expressed in the ventral diencephalon, it is expressed in the adjacent tegmentum. The expansion of Rathke's pouch could either result from the mispatterning of the ventral diencephalon/tegmentum boundary, resulting in an increase of inductive signals secreted by the ventral diencephalon, or it could result from loss of β-catenin in the neural crest derived mesenchyme, which would reveal an unappreciated role for the neural crest in regulating Rathke's pouch.

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Program/Abstract # 363
Sprouty2 restricts the progenitor field size of the mouse circumvallate papilla
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Taste is mediated by receptors that are located in the epithelial/mesenchymal structures on the tongue known as gustatory papillae. Three morphologically distinct types of gustatory papillae (fungiform, foliate, and circumvallate) are found on the dorsal surface of the tongue. Mice have a single circumvallate papilla (CVP) in the central posterior portion of the tongue. Here, we report that a null mutation for *Sprouty2* (*Spry2*), which encodes an antagonist of receptor tyrosine kinase signaling, leads to duplication of the CVP. We investigated the morphogenesis of the CVP in *Spry2* null mutant embryos and wild-type littermate controls. Between embryonic days (E) 11.5 and E14.5, the CVP developed from an epithelial placode into a bell-shaped structure with characteristic epithelial trenches. By E12.0, when the CVP placode was first morphologically detectable, the mutant placode was larger than the wild-type, and the cells appeared less condensed in the mutant. Whilst the wild-type CVP maintained a condensed circular shape throughout its morphogenesis, the mutant placode became elongated. The elongated CVP in the mutant split into two separate papillae by E14.5. Our results indicate that *Spry2* functions to ensure that the proper number of CVP forms in the embryo and that *Spry2* function is likely required from the earliest stages of CVP placode development to restrict the size of the progenitor field. Ongoing investigations are focused on determining which signaling pathway is perturbed in the CVP of *Spry2* null embryos.

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Program/Abstract # 364
Function of Sproutys in salivary gland branching morphogenesis
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In humans and mice, the salivary glands play a key role in aiding digestion, preserving the teeth from decay and providing lubrication for eating and vocalization. In mammals there are three main paired salivary glands: the submandibular, sublingual and parotid glands. The submandibular gland develops as a single epithelial bud on a primary duct that invades the underlying mesenchyme and undergoes a subsequent process of branching morphogenesis. Loss of function mutations in genes encoding FGF ligands and receptors demonstrate an important role for FGF/RTK signaling in the development of the submandibular gland. In mice, null mutations in *Fgf10*, *Fgf8* and *Fgfr2b* cause defects in salivary gland morphogenesis resulting in aplasia of the submandibular gland. In order to further study the role of RTK signaling in salivary gland development we generated null alleles of the intracellular RTK antagonists sprouty1 (*Spry1*) and sprouty2 (*Spry2*). In *Spry1*;*Spry2* compound null embryos, salivary gland development is perturbed, resulting in abnormalities of the primary duct, including ectopic end bud formation, and defects in secondary duct formation. To further characterize these defects we are performing microarray analyses to determine changes in the relative levels of gene expression in control and mutant salivary glands. The ongoing study of Sprouty function in salivary gland branching morphogenesis provides an opportunity to further elucidate the role of Sprouty genes and RTK signaling during embryonic branching morphogenesis.

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Program/Abstract # 365
Two sets of *C. elegans* mutants: The short and the amorphous pharynx phenotypes
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The *C. elegans* pharynx provides a simple model to understand the genetics cell patterning and morphogenesis. We have completed a

genetic screen for worms with abnormal pharynx muscle morphology facilitated by an integrated *myo-2GFP* reporter gene, allowing for rapid identification of animals with misshapened or missing pharynx. We have focused on two major classes of mutants, those with a short, wide pharynx, and those with amorphously shaped pharynx muscle cells. Twenty mutants manifested short and wide pharynges, suggesting that genes required for embryonic elongation of the pharynx were mutated. We have mapped many of these to small regions of chromosomes by SNP mapping. We have identified that two of the mutant lines represent alleles of sma-1, a beta-spectrin; however, many other mutant lines do not map to any previously described short-pharynx gene loci. Most of these mutant lines are larval lethal; acrylic bead feeding assays have shown that they are unable to ingest food. Further mapping will be required to identify the actual genes resulting in the phenotypes. In addition, we have identified a series of mutant lines in which the pharynx muscle cells do not appear to have normal adhesion to the pharynx. We have mapped two alleles with this phenotype to chromosome I, and shown that they do not complement each other. The acrylic bead feeding assays also suggest that the arrested larvae are not capable of ingesting food. Because of the amorphous shape of the pharynx, we are counting the number of pharynx muscle nuclei present to determine if muscle cells are missing.

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Program/Abstract # 366
Sprouty function in pancreas morphogenesis
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Two epithelial buds from the endoderm undergo a series of morphogenetic events to form the definitive mouse pancreas. To study this, the three dimensional structure of the pancreatic epithelium was examined at various stages of development using a combination of immunostaining and confocal techniques. Examination of several wild-type CD1 specimens collected between embryonic days (E)9 and E14 suggests that pancreas morphogenesis is stereotyped, allowing us to reconstruct the series of morphogenetic events that lead to the formation of the definitive pancreas. Using this system, we have begun to examine the function of Sprouty (*Spry*) genes, which encode feedback antagonists of Receptor Tyrosine Kinase (RTK)-signaling, in pancreas morphogenesis. Removing either *Spry1* or *Spry2* singly has no obvious effect, however removing combinations of these results in defects in the morphogenesis of the pancreatic epithelium. Furthermore, ectopic budding of the endoderm is observed near the region where the pancreas normally forms. The significance of these phenotypes is currently being investigated.

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Program/Abstract # 367
ENU screen for mutations in zebrafish pancreas development
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A haploid ENU (N-ethyl-N-nitrosourea) screen was performed on Ekkwill (EK) males to look for endocrine pancreas mutations by screening with *islet-1* (*isl1*). We recovered and confirmed in diploids, two pancreas mutants from our screen. 835.4 mutant embryos lack *isl1* expression in the endoderm but maintain CNS expression. Also, they lose all pancreas, liver and intestinal markers. It was found that they lose early endoderm markers such as *sox17*, *foxa2*, and *foxa1*, but have decreased *sox32* expression. As they develop, they become curved and develop a swollen pericardium by 48hpf and is embryonic lethal by day 4/5. We are currently mapping the 835.4 mutation by using CA repeat markers. 88.21 mutant embryos lack *islet-1* expression in the endoderm, but maintain CNS expression. We mapped the mutation to the catalytic domain of the *raldh2* gene where there are already 2 alleles, *neckless* (*nls*) and *no fin*. Our phenotype is similar to *nls*, in that no fin buds develop, swollen pericardium appears 3dpf and is embryonic lethal by day 4/5. Like *nls*, 88.21 mutants lose many endoderm markers, but upon further evaluation it was discovered that both *nls* and 88.21 maintain some endoderm marker expression. We injected a translational morpholino targeting *raldh2* and were able to knockdown endoderm marker expression. When we treat wildtype embryos with DEAB, an inhibitor of retinaldehyde dehydrogenases, endoderm marker expression is completely abolished. We performed qPCR and low levels of maternal *raldh2* mRNA was detected at 3hpf which may explain the residual expression seen in *nls* and 88.21.

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Program/Abstract # 369
Role of tight junction proteins in zebrafish liver morphogenesis
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Hepatocytes are the primary cells in the liver that produce bile for lipid metabolism. During liver morphogenesis, hepatocyte precursors are actively migrating, proliferating, and differentiating into hepatocytes or cholangiocytes (epithelial cells of the bile ducts). Bile duct formation also requires the polarization of hepatocytes such that their apical domain defines the bile ducts. This apical domain is defined by tight junctions. In an *in situ* hybridization expression screen in zebrafish, a member of the claudin family was found to be expressed primarily in the liver in the early stages of liver morphogenesis. Its expression becomes restricted to specialized cells of the liver at later stages. Claudins are membrane proteins that are part of the tight junctions. Their main function is to act as a paracellular transport barrier. Surprisingly, knockdown of claudin expression causes a small liver phenotype, suggesting that it plays a role in early liver morphogenesis. The role of Claudins in migrating, proliferating, and differentiating cells is unknown. They were initially identified as calcium-independent adhesion molecules mediating cellcell contacts. During early stages of liver morphogenesis, hepatic cells migrate out of the intestinal rod as a cohort of cells. One possible non-canonical role of Claudins may be to act as adhesion molecules to facilitate migration. Furthermore, recent *in vitro* evidence suggests that tight junctions communicate with the nucleus to mediate proliferation, gene expression, and differentiation depending on the cellular environment. My project aims to investigate these non-canonical roles of claudins during liver morphogenesis.

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